INDEPENDENT AND INTERACTIVE EFFECTS OF TONIC RESPIRATORY SINUS ARRYTHMIA AND NEUROTICISM ON STRESS RELATED PRESLEEP AROUSAL

by

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A thesis submitted to the faculty of
The University of Utah
in partial fulfillment of the requirements for the degree of

Master of Science

Department of Psychology

The University of Utah

August 2010

The University of Utah Graduate School

STATEMENT OF THESIS APPROVAL

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ABSTRACT

Sleep quality requires adequate inhibition of mental, behavioral, and physiological processes that perturb the sleep-wake system—a process captured by the construct presleep arousal. The extent to which individual differences in tonic high-frequency heart rate variability (or respiratory sinus arrhythmia [RSA]) and neuroticism predicted stress-related presleep arousal was examined. Eighty four healthy young adults completed measures of presleep arousal prior to and following a laboratory stressor. Neuroticism predicted stress-related changes in cognitive presleep arousal. Tonic RSA moderated the effect of neuroticism on somatic presleep arousal: Neuroticism was associated with poststress increase in somatic presleep arousal only when accompanied by low tonic RSA. These findings demonstrate that identifying individual differences in vulnerability to prolonged stress responses culminating in higher presleep arousal may be pertinent to understanding the development of chronic sleep problems.

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CHAPTER I

INTRODUCTION

Poor sleep is associated with impaired work performance, poorer social functioning, and the onset and progression of many chronic conditions, such as asthma, back pain, heart disease, and stroke (Ohayon & Shapiro, 2002). Even modest levels of sleep disruption can lead to negative outcomes such as susceptibility to infectious illness (Cohen, Doyle, Alper, Janicki-Deverts, & Turner, 2009) and to coronary artery calcification-- a precursor to atherosclerosis (King, Knutson, Rathouz, Sidney, Liu, & Lauderdale, 2008). Risk factors for the development of chronic sleep problems can be divided into predisposing factors and precipitating events (Spielman, Caruso, & Glovinsky, 1987). Research has indicated that both major life events (Bernet, Merrill, Braitwaite, VanOrden, & Joiner, 2007; Gregory, Caspi, Moffitt, & Poulton, 2006; Healy, 1981; Vahtera, Kivimaki, Hubin, Korkeila, Suominen, Paunio, et al., 2007) and daily hassles (e.g., work-related demands, arguments with a spouse) are associated with disrupted sleep (Brantly, Wagonner, Jones, & Rappaport,1987; Shaver, Johnston, Lentz, & Landis, 2002). Indeed, enhanced and/or prolonged stress responses may be an underlying mechanism for the development of insomnia in vulnerable individuals (Bastien, Vallieres, & Morin, 2004; Drake, Richardson, Roehers, Scofield, & Roth, 2004). The current study examined the independent and interactive effects of individual

difference vulnerability factors with hypothesized associations to stress-related sleepdisruption. Specifically, the extent to which individual differences in tonic high-frequency heart rate variability or respiratory sinus arrhythmia (RSA) and neuroticism predicted stress-related presleep arousal was examined.

Heightened stress-related arousal may be physiological (e.g., racing heart, muscle tension) or cognitive (e.g., rumination) in nature. Importantly, stress responses may persist long after the termination of a stressful event, a phenomenon termed prolonged activation (Pieper & Brosschot, 2005). Sleep quality can be disrupted by prolonged activation and represents an important endpoint of the stress response because it is associated with restorative processes (Uchino, Smith, Holt-Lunstad, Campo, & Reblin, 2007). Research has demonstrated that prolonged activation resulting from experimentally-induced stress serves to increase arousal thereby delaying sleep onset (Brosscht, Gerin, & Thayer, 2006; Hall, Buysee, Nowell, Nofzinger, Houck, Reynolds, Kupfer, & Thayer, 2004; Morin, Rodrigue, & Ivers, 2003). Further, cognitive and somatic arousal prior to bedtime (i.e., presleep arousal) discriminate good sleepers from poor sleepers (Gross & Borkovec, 1982).

However, the nature and directionality of the relationship between somatic and cognitive presleep arousal remains unclear. Theoretical models regarding the development of insomnia emphasize cognitive arousal as a primary predisposing factor for insomnia, but also include physiological presleep arousal as a distinct cause (Morin, 1993). Research indicates that induced physiological arousal in normal sleepers can disrupt sleep (Bonnet & Arrand, 1992; 1994;1995, Okuma, Matsuoka, Matse, &

Toyomura, 1982;Tang & Harvey, 2004) and may be associated with cognitive processes such as worry prior to bedtime (Schapkin, 2000, Bonnet, Balkin, Dinges, Roehers, Rogers, & Wesensten, 2005). In a recent study examining normal sleepers, induced physiological arousal (with caffeine) was associated with an overall increase in somatic preoccupation and intrusive thoughts about the inability to sleep (Omvik, Pallesen, Bjorvan, Thayer, & Nordhus, 2007). These findings suggest that cognitive arousal prior to bedtime may be an epiphenomenon of physiological presleep arousal. Thus, in vulnerable populations, cognitive arousal may not be the sole explanation for the development of insomnia, given that the precursor may be physiological activation prior to bed time.

Empirical evidence has implicated multiple physiological parameters contributing to sympathetic hyperarousal characteristically found in chronic sleep problems (Lichstein, Wilson, Noe, Aguillard, & Bellur, 1994; Lushington, Dawson, & Lack, 2000; Nofzinger, Norwell, & Buysee, 1999; Vgontzas, Tsigos, Bixler, Stratakis, Zachman, Kales, et al., 1998). However, such studies have generally failed to find physiological differences between individuals with insomnia and matched controls (Bastien, et al., 2008). Prior studies have focused on the measurement of sympathetic activation to the neglect of parasympathetic functioning. The effective inhibition of sympathetic nervous system responses by parasympathetic functioning may be important for maintaining stability in response to stressful events (i.e., inhibiting prolonged activation and presleep arousal) that could disrupt normal sleep (Brosschot, Van Dijk, & Thayer, 2007; Espie, 2002; Thayer & Lane, 2000). Obtaining adequate sleep quality appears to require proper

inhibition of mental, behavioral, and physiological processes that perturb the sleep-wake system (Espie, 2002). Therefore, insomnia may be characterized by the inability to readjust the sleep-wake system when situational and personal factors challenge normal sleep. Thus, identifying individual differences in vulnerability to prolonged stress responses culminating in higher presleep arousal may be pertinent to understanding the development of chronic sleep problems.

As suggested above, sustained sympathetic nervous system activity may be a potential mechanism by which stress impacts sleep. Recent models of self-regulation, most notably the Polyvagal Theory (Porges, 2007) and the Neurovisceral Integration Model (Thayer & Lane, 2000; 2009), highlight the role of the parasympathetic nervous system in exerting inhibitory control over complex physiological, behavioral, and emotional processes involved in self-regulation and adaptability. Moreover, these models emphasize the importance of inhibitory processes that are achieved via top-down processing from multiple brain structures that support aspects of attention and response inhibition that are central in executive cognitive functions (Ochsner & Gross, 2007; Posner, Rothbart, Sheese, & Tang, 2007).

Thayer and Lane (2000; 2007; 2009) suggest that the ability to flexibly engage and disengage this series of cortical and subcortical networks may have important implications for the effective regulation of health-related physiological processes, mood, and cognition. In support of these ideas, empirical evidence indicates that these regulatory processes are all associated with vagally-mediated cardiac function that can be indexed with RSA (Applehans & Leuken, 2006; Berntson, Bigger, Eckberg, Grossman,

Kaufmann, Malik, et al., 1997; Butler, Wilhelm, & Gross, 2006; Diamond & Hicks, 2005; Fabes & Eisenberg, 1997; Gyurak & Ayduk, 2008; Rottenberg, Clift, Bolden, Salomon, 2007; Rottenberg, Wilhelm, Gross, Gotlib, 2002; Smith, Cribbet, Nealey-Moore, Uchino, Williams, MacKenzie & Thayer, 2009; Thayer & Friedman, 2004). RSA can be thought of as variability in heart rate that corresponds to changes in respiration. Reduced resting or tonic RSA has been linked to glucose regulation, HPA axis functioning, inflammation, and cardiovascular disease risk (Thayer & Lane, 2007; Thayer & Sternberg, 2006).

Moreover, research indicates that disrupted sleep is associated with decreased parasympathetic nervous system functioning (Bonnet & Arand, 1998; Hall, Thayer, Germain, Moul, Vasko, Puhl et al., 2007), perhaps due to parasympathetic dominance during the restorative portion of the sleep cycle (Otzenberger, Simon, Gronfier, & Brandenberger, 1997). In patients with insomnia, RSA during sleep can serve as an index of fragmented sleep (Sfroza, Pichot, Cervena, Barthelemy, & Roche, 2007). Research conducted in healthy adults, indicates that acute stress predicts reduced RSA prior to sleep onset (Brosschot et al., 2007) and during the night (Hall, Vasko, Buysee, Ombao, Chen, Cashmere & et al., 2004). These findings suggest that poor parasympathetic nervous system functioning, as indexed by lower tonic RSA, may play a role in stress-related sleep disruption, either as an independent vulnerability factor or as a moderator of the association of other known vulnerability factors, such as neuroticism, with prolonged stress-related arousal.

The personality factor neuroticism, characterized by an increased propensity to negative affect and greater experience of daily hassles, chronic stressors and major life events (Affleck, Tennen, Urrows, & Higgins, 1994; Bolger & Zuckerman, 1995; Gunthert, Cohen, & Armeli., 1999; Magnus, Diener, Fugita, & Pavot, 1993; Suls, Green, & Hill, 1998) may constitute a particular vulnerability to prolonged stress-related arousal and associated sleep disruption (Harvey, 2002b; Vahtera, Kivimaki, Hublin, Kokeila, Suominen, Paunio, et al., 2007). There is empirical evidence that neuroticism is associated with poor sleep quality (Dorsey & Bootzin, 1997; Gray & Watson, 2002; Williams & Moroz, 2009). Further, individuals high in neuroticism exhibit more pronounced mood changes following a poor night of sleep than do individuals low in neuroticism (Blagrove & Ackenhurst, 2001).

Whereas repeated episodes of stress-related sleep disruption may place vulnerable individuals at risk for the development of chronic insomnia (Bonnet & Arrand, 2000; Drake, et al., 2004), the pathways are not well understood. Although neurotic individuals do not exhibit greater reactivity to laboratory stress, individuals high in facets of neuroticism (i.e., anxiety, hostility, and depression) experience prolonged activation following stressful events (i.e., poorer recovery; Chida & Hamer, 2008), as well as elevated blood pressure prior to sleep onset (Raikkonen, Matthews, Flory, & Owens, 1999) and during sleep (Pasic, Shaprio, Motivala, & Hui, 1998; Shapiro, Jammer, & Goldstein, 1997). Thus, individuals high in neuroticism may exhibit prolonged activation following stress exposure. Importantly, prolonged activation is thought to be characteristic of individuals with poorer parasympathetic nervous system functioning.

Specifically, when parasympathetic activity is disinhibited or withdrawn (i.e. low RSA) and sympathetic dominance occurs, the result is a pattern of affective, behavioral, and physiological imbalance that when sustained for long periods of time can lead to deleterious somatic and psychological consequences (e.g., McEwen, 1998). This type of prolonged activation can affect restorative processes such as sleep. Thus, individuals who are high in neuroticism and have low tonic RSA may be at particular risk for prolonged activation from stress, resulting in higher presleep arousal and, consequently, poor sleep quality.

The Present Study

Examining stress-related presleep arousal in normal (i.e., non-insomnia) populations is important because it may elucidate pathways by which vulnerable individuals develop chronic sleep problems. The majority of the research on stress-related sleep disruption has focused on exposure to stressful life events (Bernert & Joiner, 2007; Gregory, Caspi, Moffitt, & Poulton, 2006; Vahtera, Kivimaki, Hublin, Korkeila, Suominen, Paunio, et al., 2007). The present study examined individual differences in vulnerability to stress-related presleep arousal using a laboratory stress paradigm. Specifically, this study examined the associations among tonic RSA, neuroticism, and stress-related changes in presleep arousal. The extent to which tonic RSA, an index of parasympathetic nervous system functioning, moderates the effects of neuroticism on stress-related presleep arousal was examined. It was predicted that higher neuroticism would be more strongly associated with stress-related increases in presleep arousal when accompanied by low tonic RSA.

CHAPTER II

METHOD

Participants

Eighty-four participants (52 % male), enrolled in introductory psychology courses, completed the current study for course credit. The mean age was 22.9 years, SD = 5.73. The sample was 80 % Caucasian. Exclusionary criteria included medication known to influence cardiac functioning (i.e., beta blockers, antihistamines). Participants were required to refrain from caffeine consumption, use of nicotine, and exercise two hours prior to the beginning of the experiment. Compliance with these criteria was assessed prior to participation in the laboratory session.

Measures

Social Competence Interview (SCI; Ewert, Jorgensen, Suchday, Chen, & Matthews, 2004). The SCI is a semistructured interview designed to assess socio-emotional responses to a real-life event. The interview is structured such that the first 4 to 6 minutes are devoted to "re-living" the stressful event (specific people, places, what the participant said and felt). The interviewer (consistent with prior research, all interviewers were female) was instructed to help the person relive the event without challenging or harassing him or her. The remainder of the interview was focused on coping and the participant's preferred resolution to the problem ("pretend that you were making a movie

about the event we just discussed and you could make it end any way you want"), strategies for achieving a desired outcome, and confidence in obtaining that desired outcome. The SCI has demonstrated good internal consistency, validity, and temporal stability over a three month period, provoking stress responses comparable to or at higher levels than tasks such as mental arithmetic or mirror tracing, and showing stronger associations to ambulatory blood pressure than those stressor tasks (Ewert, Jorgensen, Suchday, Chen, & Matthews, 2004).

NEO Personality Inventory-Revised (NEO PI-R; Costa & McCrae, 1992). The NEO PI-R is a self-report measure used to assess five higher-order factors of personality, each with six lower-order facet scales: Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness. The higher-order factors and the facet scales have demonstrated high internal consistency, and convergent and discriminate validity (Costa & McCrae, 1992), and longitudinal stability in nonpsychiatric populations (Costa, Herbst McCrae, & Siegler, 2000). The focus of the current study was on the 48-item neuroticism scale.

The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) The PANAS is a self-report measure designed to assess both positive affect (PA; e.g., excited, inspired, determined, active) and negative affect (NA; e.g., afraid, nervous, guilty, ashamed). Participants were asked "to what extent did you experience each of the following adjectives in the moment?" Responses were made on a Likert scale ranging from 1 (very slightly or not at all) to 5 (extremely). The PANAS has demonstrated good internal consistency (PA scale alpha range from .86 to .90, NA scale alpha range from .84

to .87), test-retest reliability (for momentary reports of mood PA = .54, NA = .45), and good convergent validity with other mood measures. Subsequent studies have confirmed the latent structure (robust comparative fit index = .94) of two correlated factors, positive affect and negative affect (Crawford & Henry, 2004).

Presleep Arousal Scale (PSAS; Nicassio, Mendlowtiz, Fussell, & Petras, 1985). Prior to and following the laboratory session, participants completed the PSAS. The PSAS is a self-report measure containing 16 items that assess both cognitive (e.g., racing thoughts, worries) and somatic (e.g., heart racing, muscle tension) states of arousal at bedtime. For each item, participants are instructed to indicate how intensely each of the symptoms are experienced when attempting to fall asleep, with scores ranging from 1 (not at all) to 5 (extremely), yielding cognitive and somatic arousal subscales, each ranging from 8-50. Higher scores reflect the perception of greater intrusive cognitions and physiological arousal at bedtime. This scale has demonstrated adequate internal consistency (alphas = .67 to .88 for cognitive and somatic subscales, respectively) and has been shown to reliably discriminate normal sleepers from those with insomnia (Morin & Espie, 2003).

Heart rate and respiration. The electrocardiogram (ECG) data were collected from participants using three spot electrodes placed in the standard lead II configuration. The ECG was digitized at 1000Hz, measured using a Biopac MP100 system, (ECG100, GSR100, and RSP100 modules; Biopac Systems Inc. Santa Barbara, CA) and collected using Acqknowledge Software (Biopac Systems Inc.). Respiration was measured by placing a respiration band at the xyphisternal junction.

Respiratory sinus arrythmia (RSA). The raw ECG data were first inspected using automated software and then visually inspected according to the guidelines for detecting artifacts and abnormal beats (Berntson, 1990). ECG was measured continuously during all experimental sessions. There are two basic approaches to quantifying heart rate variability: 1) the use of descriptive statistics to characterize heart periods and 2) the extraction of the variance of periodic components of heart rate at a specific frequency (spectral analysis). For the purpose of the current study, HRV Analysis Software 5.2 (Mindware Cardiography system, Gahanna, Oh) was used to verify, edit, and summarize cardiovascular data. For each participant, ECG data were ensemble averaged for each minute. The same HRV analysis software was used to derive heart rate variability (ms^2/Hz) by applying spectral analysis to the interbeat interval series (IBI series – the time between successive R-peaks) from the ECG. The IBI series was time sampled at (4Hz) to produce an equal time series. Time series analysis of IBI series using spectral approaches assumes that data points are equally spaced. Successive IBI series are spaced unevenly in time, and thus must be subjected to methods to derive an equal time series. This problem is intensified over time and by laboratory stressors. Berntson, Bigger, Eckberg, Grossman, Kaufman, Malik, et al. (1997) suggest a sampling rate of 4Hz for capturing HF heart rhythms and for generating an equally spaced IBI series. The equal time series was then detrended, end tapered, and submitted to a fast Fourier Transformation according to procedures outlined by Bernston, et al. (1997). The detrending process involves the application of a second order polynomial to the heart rate time series. Spectral analysis was used to decompose heart rate variability at specific

frequency components. The frequency component of RSA is the parasympathetically-driven oscillations that corresponded to the High Frequency (HF) portion of the respiration cycle (0.15-0.40Hz). Respiratory rates that fell below the cutoff point (0.15Hz) were identified by examining the frequency band, and data from that minute were excluded.

Procedure

Following informed consent, participants were screened for exclusionary criteria and for compliance with pre-experimental requirements in individual laboratory sessions. Questionnaires were completed that assessed participant's presleep arousal and sleep quality from the prior night. Participants were instructed to relax quietly while breathing normally for a 10-minute resting baseline period, during which ECG and blood pressure readings were collected. At the end of baseline, participants completed a measure of state positive and negative affect. Following the baseline period, participants were instructed to indicate their most distressing experiences over the last several months, by rank ordering a list of stressors (derived from the Inventory of College Students' Recent Life Experiences; Kohn, Lafreniere, & Gurevich, 1990) commonly experienced by college students. One of the participant's top-ranked stressors was selected for discussion using the Social Competence Interview (SCI; Ewart, Jorgensen, Suchday, Chen, & Matthews, 2004) (described below). Immediately following the stressor period, participants provided ratings related to the prior task (difficulty, engagement, stressfulness, NA, and PA) while resting quietly. Participants were provided instructions about completing an online questionnaire of presleep arousal, for that evening following the laboratory stress

task. A reminder email was sent in the afternoon or early evening to prompt participants about the on-line survey.

CHAPTER III

RESULTS

Descriptive and Correlational Statistics

Means, standard deviations, and zero-order correlations among neuroticism, tonic RSA, and both cognitive and somatic presleep arousal (including prior night and poststress ratings) are presented in Table 1. The sample mean corresponds to a T-score of 49 for published NEO-PI-R College Norms averaged for males and females (M = 50, SD = 10; Costa & McCrae, 1992), indicative of Average neuroticism. Neuroticism was significantly correlated with both prior night and poststress cognitive and somatic aspects of presleep arousal. Tonic RSA was not significantly correlated with any index of presleep arousal, ps>.05.

Manipulation Check: Stress Related Presleep Arousal and Affective Responses

Subjective ratings of positive and negative affect (PA and NA, respectively) were collected during the laboratory session following baseline and following the discussion of a stressful event. Paired samples t-tests demonstrated that ratings of negative affect, M = 15.15, SD = 4.61, were significantly higher than baseline, M = 14.03, SD = 4.64, t = 2.82, p<.05, and that ratings of positive affect, M =25.31, SD=7.8, were significantly lower than baseline, M = 27.24, SD = 6.77, t = 2.82, t= -3.95, p<.0001. Thus, the SCI resulted in

Table 1.

Zero Order Correlations Among Study Variables.

Variable	1	2	3	4	5	6
1. Neuroticism	_	**0.28**	**0.38**	**0.25**	**0.44**	-0.02
2. Prior Night Somatic PSA		_	**0.54**	**0.69**	**0.41**	0.15
3. Prior Night Cognitive PSA			_	**0.41**	**0.63**	0.09
4. Poststress Somatic PSA				_	**0.47**	-0.02
5. Poststress Cognitive PSA					_	0.12
6. Tonic RSA						_
Mean	92.1	9.8	14.3	10	17.2	6.69
Standard Deviation	25.9	3.28	6.45	3.35	6.15	1.18

Note: Correlations in boldface indicate $p<.05$. PSA=presleep arousal; RSA=respiratory sinus arrhythmia.

increased NA and decreased PA. Central to the focus of the study, a paired samples t-test demonstrated that ratings of poststress presleep arousal, $M = 27.2$, $SD = 8.28$, was significantly higher than the prior night presleep arousal rating, $M = 24.2$, $SD = 8.68$, $t = 3.82$, $p < .001$, indicating that the laboratory stressor served to increase presleep arousal across participants.

Regression Analyses

Regression analyses were conducted to examine the independent effects of tonic RSA and neuroticism on stress-related change in presleep arousal, as well as the hypothesized moderation models. Separate regression analyses were conducted that examined the independent effects of tonic RSA and neuroticism predicting poststress cognitive and somatic presleep arousal, controlling for prior night's presleep arousal. To examine the moderating effect of tonic RSA on the association between neuroticism and stress-related changes in presleep arousal, separate regression models for somatic and cognitive presleep arousal that included the first order effects and the RSA x neuroticism interaction term were examined. In order to establish the specificity of effects on *change* in presleep arousal, regression models were also conducted to examine prior night presleep arousal. In all regression models the variables were centered to minimize multicollinearity and standardized prior to analyses. Standardized betas are presented.

Prior Night Presleep Arousal

Tonic RSA did not independently predict prior night ratings of cognitive, $\beta = .09$, $p > .05$, or somatic, $\beta = .14$, $p > .05$, presleep arousal. Neuroticism independently predicted

prior night cognitive, β = .38, p <.0001, and somatic β = .28, *p* >.01 ratings of presleep arousal. However, regression analyses indicated that the RSA x neuroticism interaction did not predict prior night ratings of cognitive, β = .05, *p* >.05, or somatic presleep arousal, β = .09, *p* >.05.

Stress Related Presleep Arousal

Tonic RSA did not independently predict either poststress somatic, β = .14 *p* > .05, or cognitive, β = .09, *p* >.05, presleep arousal, controlling for prior night ratings of presleep arousal. Neuroticism significantly predicted poststress cognitive aspects of presleep arousal controlling for prior night cognitive aspects of presleep arousal, β = .21, *p*<.05, but did not independently predict stress-related change in somatic presleep arousal, β = .06, *p*>.05.

In regressions examining the association between neuroticism and stress-related changes in presleep arousal that included the RSA x neuroticism interaction, the interaction was significant for stress-related increases in somatic presleep arousal, β = -.31, *p*<.001, ΔR^2 = .07, but not changes in cognitive presleep arousal, β = -.06, *p*>.05. In order to probe the significant interaction, the regression model was restructured on high and low values (one standard deviation above and below the mean) of tonic RSA (Cohen, Cohen, West, & Aiken, 2003). Neuroticism was related to stress-related increases in somatic presleep arousal under conditions of low tonic RSA, β = .42, *p*<.01, but not under conditions of high tonic RSA, β = -.22, *p*>.05 (see Figure 1).

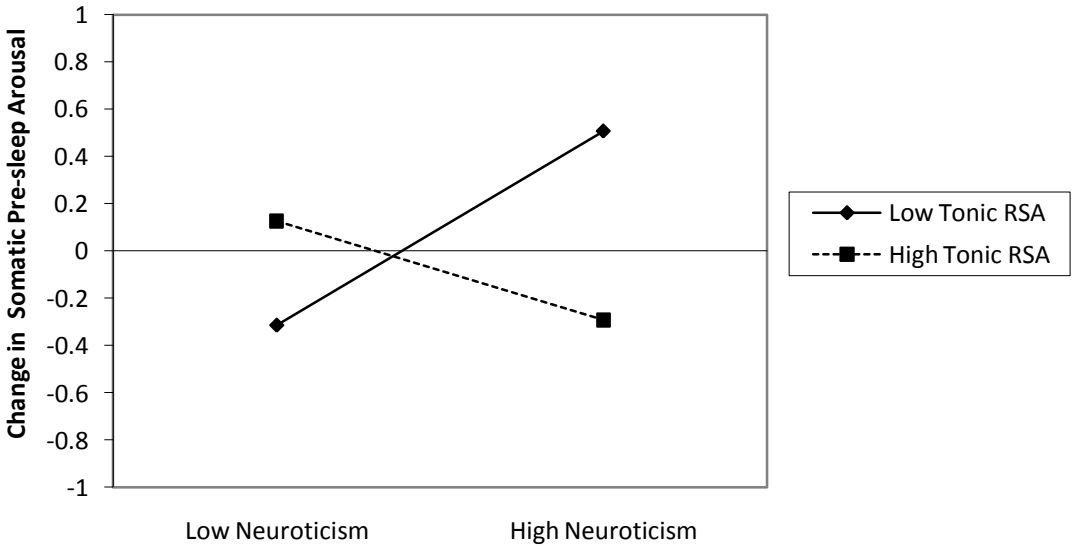

Figure 1.

Plot of predicted values (z-scores) for two way interaction between neuroticism and tonic RSA predicting change in somatic aspects of presleep arousal.

Neuroticism Facet Level Analyses

In order to understand which aspects of neuroticism were associated with stress-related somatic presleep arousal, the individual facets were examined. The Depression facet was associated with poststress cognitive presleep arousal controlling for initial levels of cognitive aspects of presleep arousal, β = .22, p<.05. No other facets of neuroticism were associated with stress-related change in somatic or cognitive presleep arousal, *ps* >.05. In regressions examining the association between neuroticism facets and stress-related changes in presleep arousal that included the RSA x facet interaction term, tonic RSA moderated the relation between the Anxiety, β = -.27, p<.01, ΔR^2 = .05, Depression, β = -0.16, p<.001, ΔR^2 = .07, Self-Consciousness, β = -.31, p<.01, ΔR^2 = .06, Impulsiveness, β = -.21, p<.05, ΔR^2 = .03, and Vulnerability, β = -.35, p<.01, ΔR^2 = .06, facets and stress-related increases in somatic presleep arousal. Anxiety, Depression, Self-Consciousness, and Impulsiveness were associated with stress-related increases in somatic presleep arousal under conditions of low tonic RSA (β = .30, β = .23, β = .41, β = .21, *p*s<.01, respectively) but not under conditions of high tonic RSA, *p*s>.05. Vulnerability was positively associated with somatic presleep arousal when accompanied by low tonic RSA, β = .38, p<.01, but negatively associated when accompanied by high tonic RSA, β = -.33, p<.05.

CHAPTER IV

DISCUSSION

The present study examined the unique and interactive effects of hypothesized vulnerability factors, tonic RSA and neuroticism, to stress-related presleep arousal. The current study adds to prior research which has demonstrated that naturally occurring stress can increase arousal prior to bedtime (Bonnet & Arrand, 1995; Gross & Borkovec, 1982; Morin et al., 2003). Among normal sleepers, stress-related presleep arousal may be an important factor leading to the development of more chronically disrupted sleep. These results can be placed within a larger theoretical framework regarding the central role of the ability to inhibit the arousal caused by situational and personal factors that challenge normal sleep (Espie, 2002).

Although stress-related arousal remains a primary candidate for the pathophysiology of insomnia, few studies have investigated individual difference vulnerability factors in healthy (i.e., noninsomnia) samples. Prior research indicates that neuroticism is a vulnerability factor associated with insomnia (Harvey, 2002b). Among healthy individuals, those high in neuroticism are more likely to report poor sleep quality (Gray & Watson, 2002) and negative daytime consequences associated with poor sleep (Williams & Moroz, 2009). In the present study neuroticism predicted stress-related increases in cognitive arousal. These findings suggest that neuroticism, a trait associated with propensity to negative affect and poor stress coping, may confer an independent risk

for stress-related presleep arousal. Facet-level analyses indicated that this effect was driven by the association of the Depression facet of neuroticism with cognitive presleep arousal. Thus, it is likely that the propensity to experience depressive mood in response to stress, and subsequent cognitive arousal prior to bedtime could be a potential mechanism linking personality to the development of more chronic sleep problems, as well as depression.

A large epidemiological study demonstrated that low tonic RSA is a risk factor for all-cause morbidity and mortality (Liao, Carnethon, Evans, Cascio, & Heiss, 2002). It was hypothesized that tonic RSA would be associated with stress-related presleep arousal in healthy individuals. Tonic RSA did not independently predict stress-related changes in somatic or cognitive presleep arousal. These findings suggest that, in healthy young adults, individual differences in tonic RSA may be most important for individuals prone to experience negative emotions.

In the present study the personality factor neuroticism predicted stress-related increases in somatic arousal only when accompanied by lower tonic RSA (i.e., poorer parasympathetic nervous system functioning). Because this interaction was not significant for ratings of the prior night's presleep arousal, this effect appears to be specific to stress responses. Although both somatic and cognitive presleep arousal appear to be important in the onset of stress-related sleep problems, prior research suggests that somatic or physiological arousal may be a precursor to, rather than a consequence of, worry (Borkovec, 1979, Freedman & Sattler, 1982). Laboratory studies have demonstrated that sleep disturbances mimicking insomnia can be induced in normal

sleepers by the administration of caffeine (Bonnet & Arrand, 1992; 1994) or a pharmaceutical stimulant that increases somatic arousal (Okuma, Matsuoka, Matse, & Toyomura, 1982; Tang & Harvey, 2004). Experimentally-induced physiological arousal through caffeine administration caused an increase in nocturnal worry prior to bedtime among normal sleepers (Omvik et al., 2007). Taken together the results from these studies suggest that physiological arousal may have a causal role in disrupted sleep by leading to cognitive activation characteristic of more chronic sleep problems.

The findings of the current study provide evidence for the interactive nature of individual vulnerability factors in predicting stress-related presleep arousal in healthy participants. Prior research has primarily examined these individual difference factors in relation to stress exposure and reactivity. The current findings highlight the potential importance of these vulnerabilities for prolonged stress-related activation and, by extension, stress-related sleep disruption. Poor sleep can inhibit restorative processes (e.g., wound healing) and exact excessive wear and tear on the body (McEwen, 1998). The inability to effectively inhibit a variety of stress-related behavioral, emotional, and physiological processes may be exacerbated by poor sleep, thereby placing vulnerable individuals at risk for developing anxiety and depression. Importantly, there is evidence to suggest that disrupted sleep precedes the onset of depression (Johnson, Roth, & Breslau, 2006; Ohayon & Roth, 2003), and that difficulties with sleep onset are associated with anxiety (Taylor, Lcihstein, Durrence, Reidel, & Bush, 2005). Relationships among psychological stress, neuroticism, tonic RSA, and sleep may be

important in sleep disorders comorbid with psychiatric conditions in which stressors may both precipitate and maintain clinically significant sleep disruption.

Limitations and Future Directions

The current sample appeared to be representative of the population from which it was drawn, but it will be important to extend these findings to other age and ethnic groups. Importantly, the sample was comprised of healthy individuals and generalizations to clinical populations (e.g., individuals with insomnia or mood disorders) should be made with caution. The current study examined short-term associations between stress and presleep arousal; thus, examining the relation between these individual vulnerability factors over a longer timeframe may further elucidate mechanisms by which vulnerable individuals develop disrupted sleep. The study also focused on subjective perceptions of physiological and cognitive arousal. Although these perceptions are central to the development of sleep disorders (Harvey, 2002a), future research should examine objective indicators of physiological presleep arousal.

These findings may have important implications for preventive intervention and, possibly, insomnia treatment. Research has indicated that among individuals with anxiety disorders, heart rate variability (HRV) biofeedback effectively reduced anxiety, anger, and sleep onset latency (Reiner, 2008). Reportedly, these effects were attained through resonant breathing and cognitive focus that led to an increase in RSA-restoring autonomic nervous system balance (Berntson & Cacioppo, 2004). HRV biofeedback has proven effective in other populations such as in individuals with asthma (Leher Vaschillo, Vaschillo, Lu, Scardella, Siddique, et al., 2004) and chronic pain (Hassett

Radvanski, Vaschillo, Vaschillo, Sigal, Karavidas, et al., 2007). Applied to vulnerable individuals, HRV biofeedback could be an effective prevention strategy aimed at reducing physiological arousal characteristic of sleep disturbance.

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